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The redox state of plastoquinone pool regulates state transitions via cytochrome b_6f complex in *Synechocystis* sp. PCC 6803

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Abstract The effects of benzoquinone analogues, phenyl-1,4-benzoquinone (PBQ) and 2,5-dibromo-3-methyl-6-isopropyl-1,4-benzoquinone (DBMIB), on state transitions in *Synechocystis* sp. PCC 6803 were investigated. PBQ induced a transition from state 2 to state 1 in the absence of actinic light whereas DBMIB caused a state 2 transition. 3-(3,4-Dichlorophenyl)-1,1-dimethyl urea could not eliminate the effects of PBQ and DBMIB. These results imply that the redox state of the plastoquinone pool controls the state transitions in vivo and cytochrome b_6f complex is involved in this process. As a working hypothesis, we propose that the occupancy of the quinol oxidation site and the movement of the Rieske protein may be pivotal in this regulation.

Key words: State transition; Plastoquinone; Cytochrome b_6f; Cyanobacteria

1. Introduction

In cyanobacteria, phycobilisomes (PBS) serve as the major light-harvesting antenna transferring energy to the photosystem (PS) II and PS I reaction centers [1,2]. Cyanobacteria are able to regulate redistribution of the excitation energy from the PBS to the two PSs and ensure that electron transport through PS I and PS II is balanced in different light regimes. This regulatory mechanism is known as state transition [3–5]. Illuminated with light absorbed preferentially by PS I (light 1), the cells shift toward state 1 and most of the energy absorbed by PBS directs to PS II, with an increase of the PS II fluorescence yield. Under light absorbed predominantly by PBS (light 2), the cells tend to shift toward state 2 and the PBS mainly transfer the excitation energy to PS I [6], with a relative decrease of the PS II fluorescence yield.

In high plant and green algae, state transitions involve the reversible phosphorylation of the light-harvesting chlorophyll (Chl) *a/b* protein complex (LHC II) [7,8], which induces the migration of LHC II between the grana (PS II-enriched) and the stroma (PS I-enriched) domains of the thykoids and therefore regulates the LHC II association with PSs (for reviews see [9,10]). The protein kinase for LHC II phosphorylation is located in the thylakoid membrane, and its activation requires both the reduction of the plastoquinone (PQ) pool [7] and the presence of cytochrome b_6f complexes [11,12]. When PQH_2 binds to the quinol oxidation (Qo) site of the cytochrome b_6f, the kinase is activated [13–15]. The dephosphorylation of LHC II results from a permanently active phosphatase [16].

By contrast, the regulatory mechanism for state transitions in cyanobacteria is not well characterized. There is strong evidence that the state transitions are regulated by the redox status of an intermediate electron carrier between PS II and PS I [17–20]. The reduction of this electron carrier induces a shift toward state 2 whereas the oxidization results in a state 1 transition. This electron carrier may be PQ or a closely associated electron carrier [19]. The distribution of excitation energy from PBS to PS I and PS II changes in state transitions but its biophysical basis remains incompletely understood. It was reported that PBS diffused quite rapidly along the surface of thylakoid membranes and the lateral diffusion of PBS could be involved in state transitions [21]. Furthermore, Bald et al. proposed that the processes of dynamic coupling and uncoupling of PBS to PS I and PS II were analogous to the mechanisms which regulated the LHC II association with PSs in chloroplasts [22].

In this work, we investigated the effects of two benzoquinone analogues, phenyl-1,4-benzoquinone (PBQ) and 2,5-dibromo-3-methyl-6-isopropyl-1,4-benzoquinone (DBMIB), on state transitions in the cyanobacterium *Synechocystis* sp. PCC 6803 by measurements of modulated Chl fluorescence and low-temperature fluorescence emission spectroscopy. Our results imply that the redox state of the PQ pool regulates state transitions via the cytochrome b_6f complex in cyanobacteria.

2. Materials and methods

The wild-type strain of *Synechocystis* sp. PCC 6803 was cultured photoautotrophically at 30°C in BG-11 medium supplied with 20 mM HEPES/NaOH (pH 7.5). The cells were grown in continuous light at 50 μE m^{-2} s^{-1}. Growth of the *Synechocystis* sp. PCC 6803 was fol-

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Abbreviations: Chl, chlorophyll; DBMIB, 2,5-dibromo-3-methyl-6-isopropyl-1,4-benzoquinone; DCMU, 3-(3,4-dichlorophenyl)-1,1-dimethyl urea; Fm, maximum fluorescence, where all Q_A is reduced and all non-photochemical quenching processes are at a minimum (e.g. in the presence of either DCMU plus actinic light or far-red light); Fm′, fluorescence intensity with all Q_A reduced under other conditions; PS I, photosystem I; PS II, photosystem II; PBQ, phenyl-1,4-benzoquinone; PBS, phycobilisomes; PQ, plastoquinone; PQH_2, plastoquinol

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lowed by monitoring the optical density at 730 nm. Cells were harvested at a OD_{730} of 0.8–1.2 by centrifugation at 6000×*g* for 7 min and resuspended in the fresh medium to a Chl *a* concentration of 5 μM. The concentration of Chl *a* was determined from the 663 nm absorption value [23].

All reagents were added from stock solutions in dimethyl sulfoxide (DMSO). The final concentrations of DMSO were lower than 0.5% (v/v), which did not cause any obvious changes of Chl fluorescence.

The yield of Chl fluorescence was continuously monitored with a PAM Chl fluorometer (Walz, Effelrich, Germany) adapted to a DW2 cuvette of Hansatech oxygen electrode at 25°C. Cells were adapted to darkness for 10 min before measurements. The intensity of modulated measurement light was lower than 0.2 μE m^{-2} s^{-1} to avoid photosynthetic electron transport [5]. Maximum fluorescence level (Fm or Fm′) was determined using saturating light pulses (3200 μE m^{-2} s^{-1}, 400 ms duration) which was produced by a KL-1500 lamp (Schott, Germany). The intensity of far-red light (>700 nm, through a homemade filter) was 80 μE m^{-2} s^{-1}. White light (200 μE m^{-2} s^{-1}) was used as actinic light in the presence of 3-(3,4-dichlorophenyl)-1,1-dimethyl urea (DCMU).

77 K fluorescence emission spectra were recorded on a Hitachi F-4500 fluorescence spectrophotometer. In the dark, reagents were added and cells were incubated for an additional 5 min. Then the samples were frozen in liquid nitrogen without glycerol to prevent functional uncoupling of the PBS from thylakoid components [24]. Excitation was performed at 580 nm for phycobilin excitation. Both the slitwidths for the excitation and emission monochromators were 5 nm.

3. Results and discussion

In the dark, cyanobacteria are normally in state 2 due to respiratory electron flow [17]. Fig. 1A shows that the dark-adapted cells displayed a low Fm′ level, which is the characteristic of state 2. When far-red light (light 1) was applied, Fm′ rose to a higher level and Fm was obtained. This is the characteristic of state 1 [20,25]. Removal of the far-red light induced a large quenching of Fm′ and a shift from state 1 to state 2.

PBQ is a commonly used electron acceptor in the measurements of PS II oxygen-evolution activity, which can bind to the Q_B site of PS II and efficiently accept electron from Q_A^- [26]. The mid-point potential of PBQ is 279 mV [27], higher than that of PQ (60 mV). Therefore, PBQ could also directly obtain electrons from the reduced PQ pool. In the absence of actinic light, addition of 50 μM PBQ led to a significant increase in the Fm′ level (Fig. 1B), which was similar to the change induced by far-red light (Fig. 1A). This indicates that PBQ addition caused a shift from state 2 to state 1. As shown in Fig. 1C, the effect of PBQ on Fm′ was in a concentration-dependent manner. Differences in the amplitude and in the kinetics of state transitions were observed at various PBQ concentrations. When the concentration of PBQ was increased from 5 to 150 μM, both the level and rise rate of Fm′ increased accordingly.

The effect of PBQ on the state transitions in *Synechocystis* PCC 6803 was further investigated by low-temperature fluorescence measurements. Fig. 2 shows the 77 K fluorescence emission spectra of the dark-adapted cells incubated in various concentrations of PBQ. The peak at 645–665 nm derives from phycocyanin and allophycocyanin. The fluorescence emission around 695 nm mainly results from the antenna CP 47 and the reaction center of PS II whereas the peak at 720 nm originates from PS I [28,29]. PBQ led significant increases in the value of the F_{695}/F_{720} ratio, in a concentration-dependent manner. This indicates that PBQ facilitated redistribution of excitation energy more in favor of PS II [25].

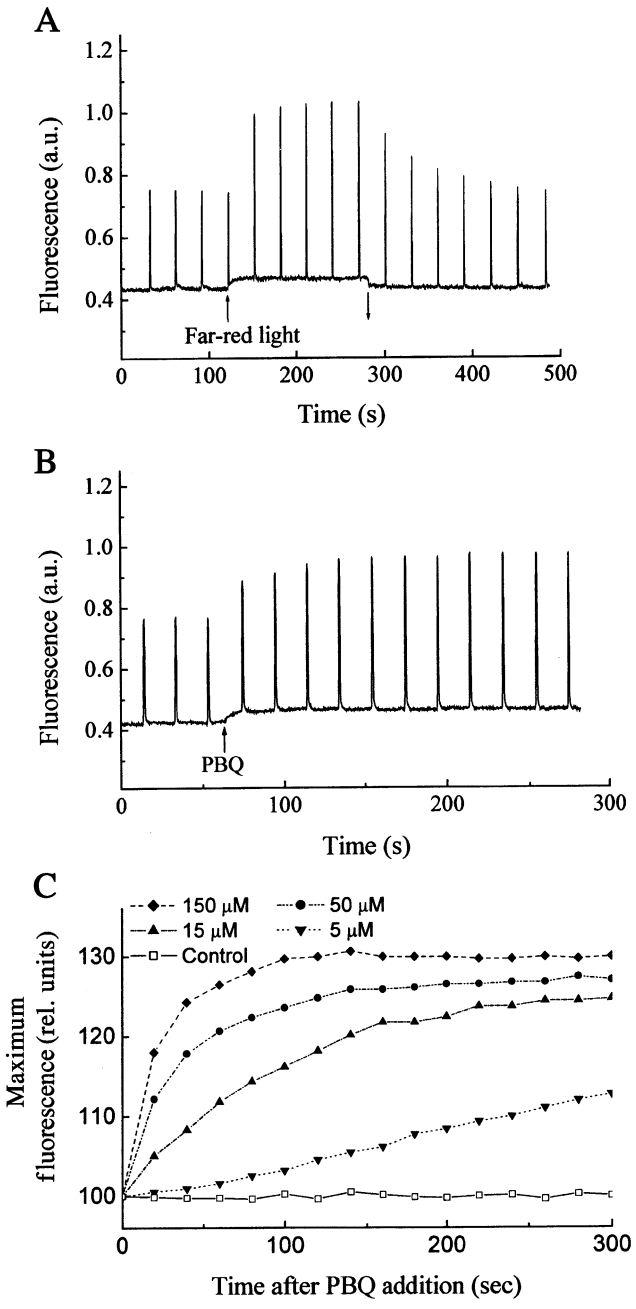

Fig. 1. Effects of far-red light or PBQ on Chl fluorescence yield in dark-adapted *Synechocystis* PCC 6803. A: Far-red light induced a transition from state 2 to state 1. The upward arrow indicates start of far-red light, and the downward arrow shows termination of the light. B: 50 μM PBQ induced a rise of Fm′ in the dark. The arrow indicates the PBQ addition. C: The effects of various concentrations of PBQ on Fm′.

These results further conform that PBQ can cause a state 1 transition in *Synechocystis* PCC 6803.

It is known that DBMIB binds to the Qo site of the cytochrome b_6f complex and blocks the transfer of electrons from the PQ pool to the cytochrome b_6f complex [30,31]. DBMIB can also bind at Q_B site of the PS II reaction center, acting as a DCMU-type inhibitor [26,32,33]. It should be noted that the mid-point potential of DBMIB is 170 mV [26], higher than that of PQ. Both DBMIB and PBQ can accept electrons from PQ and be reduced in thylakoids under light [34]. However,

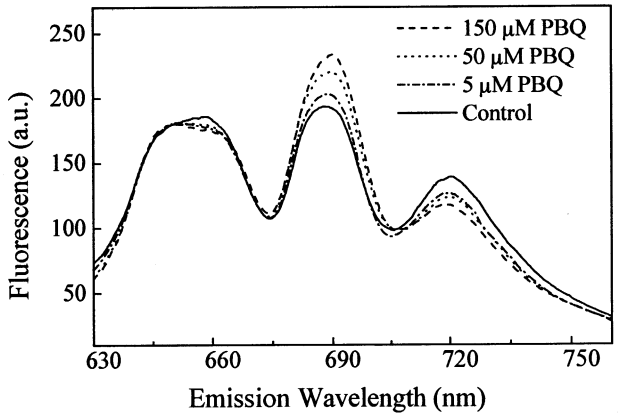

Fig. 2. Fluorescence emission spectra at 77 K of dark-adapted *Synechocystis* PCC 6803 treated with various concentrations of PBQ. The excitation wavelength was 580 nm. No glycerol was added to the cells. The spectra are normalized at 650 nm.

DBMIB did not cause a remarkable change of Fm′ in the dark-adapted cells. Moreover, it prevented far-red light from inducing an increase in the Fm′ level (data not shown). This is consistent with the observation that DBMIB inhibits the light-induced state 1 transition in *Synechococcus* 6301 [19]. Fig. 3A shows that DBMIB also suppressed the state 1 transition induced by 50 μM PBQ. With the increased concentrations of DBMIB, the rate and amplitude of the state transitions decreased gradually. When DBMIB and PBQ were at the same concentration, the cells were substantially in state 2. Furthermore, DBMIB induced an obvious decrease of Fm′ in PBQ-treated cells (Fig. 3B). El Bissati et al. reported that the presence of DBMIB caused a large quenching of Fm′ in the *Synechocystis* PCC 6803 cells under blue light (light 1) [25]. These results indicate that DBMIB can induce a state 2 transition in cyanobacteria, even in the cells already in state 1.

Since both PBQ and DBMIB can bind at the Q_B site of PS II [26], their effects on state transitions might originate from their interactions with PS II, with subsequent effects on the coupling between PS II and PBS. To test this possibility, we investigated the effects of DCMU on Fm′ in the absence or presence of DBMIB and PBQ. The presence of DCMU, with higher affinity at the same Q_B site, prevented the interaction between quinones and PS II [26]. Under actinic light, DCMU addition caused the closure of all PS II centers and immediately resulted in a rapid rise of Chl fluorescence yield, then a slower rise phase (Fig. 4A). At the end of the rapid rise phase, the fluorescence level was equivalent to the fluorescence peak Fm′ caused by a brief saturating light pulse in dark-adapted cells. After the slow phase, the fluorescence yield was similar to the Fm level obtained under far-red light illumination, indicating a shift towards state 1. However, the presence of DCMU did not affect the fluorescence yield in the DBMIB-treated cells (Fig. 4B). Furthermore, Fig. 4C shows that the presence of 5 μM DBMIB and 50 μM PBQ induced a small amplitude of state 1 transition, and the subsequent addition of DCMU plus actinic light did not change the Fm′ level. All of these results indicate that DCMU could not reverse the effects of DBMIB and PBQ, and exclude the possibility that the effects of DBMIB and PBQ on state transitions resulted from their interaction with the PS II reaction center.

According to the previous studies, state transitions in cyanobacteria are controlled by the redox state of an electron carrier intermediate between the two PSs [17–20]. The photosynthetic electron transport chain between PS II and PS I includes PQ, plastocyanin (or cytochrome c_{553}) and the cytochrome b_6f complex. There are two electron paths within the cytochrome b_6f: one is a high-potential chain, composed of the iron–sulfur cluster of the Rieske protein and the heme of the cytochrome f; the other is a low-potential chain, composed of the two hemes of the b-type cytochrome, the low-potential b_L and high-potential b_H hemes. In dark-adapted *Synechocystis* PCC 6803, all these electronic carriers were reduced [35].

In thylakoid membrane, the molar ratio of PQ to Chl a is 1:54 [36]. PBQ and DBMIB were added in large excess over the endogenous PQ molecules in our experiments. Therefore, most of the PQ molecules were oxidized upon the addition of PBQ or DBMIB. The two hemes of cytochrome b, which can reduce PQ, also were oxidized in the presence of PBQ. The fact that PBQ induced a state 1 transition implies that PQ or the b hemes may be involved in the state transitions, and supports the model which proposes that the redox state of PQ or a closely associated electron carrier regulates the state transitions in cyanobacteria [19]. However, Fig. 3B shows that DBMIB resulted in a transition from state 1 to state 2 in PBQ-treated cells, where both PQ and the b hemes were in their oxidized forms. On the other hand, the high-potential path components of the cytochrome b_6f and the plastocyanin

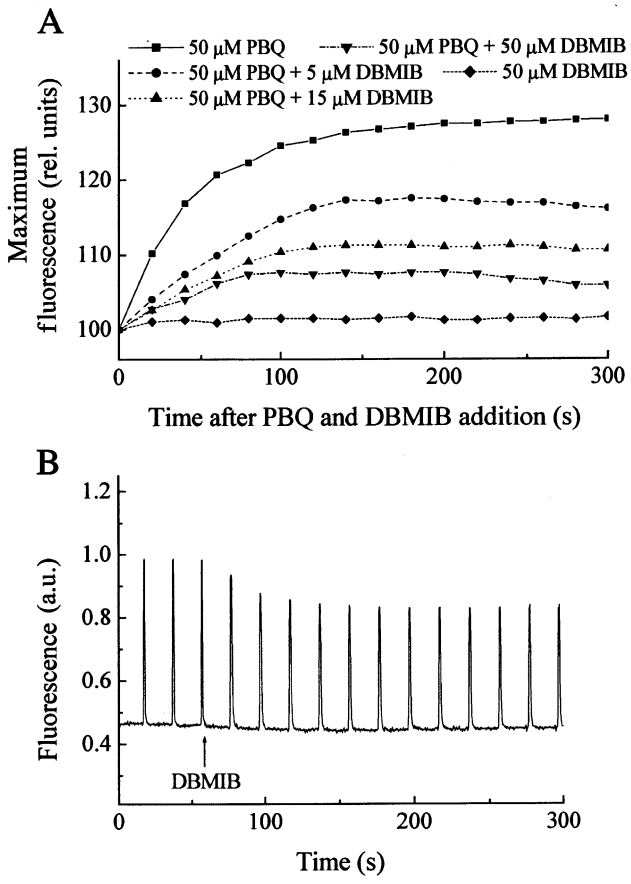

Fig. 3. Effects of DBMIB on state transitions in dark-adapted *Synechocystis* PCC 6803. A: DBMIB suppressed the state 1 transition induced by PBQ. B: 50 μM DBMIB induced a shift from state 1 to state 2 in the cells treated by 50 μM PBQ.

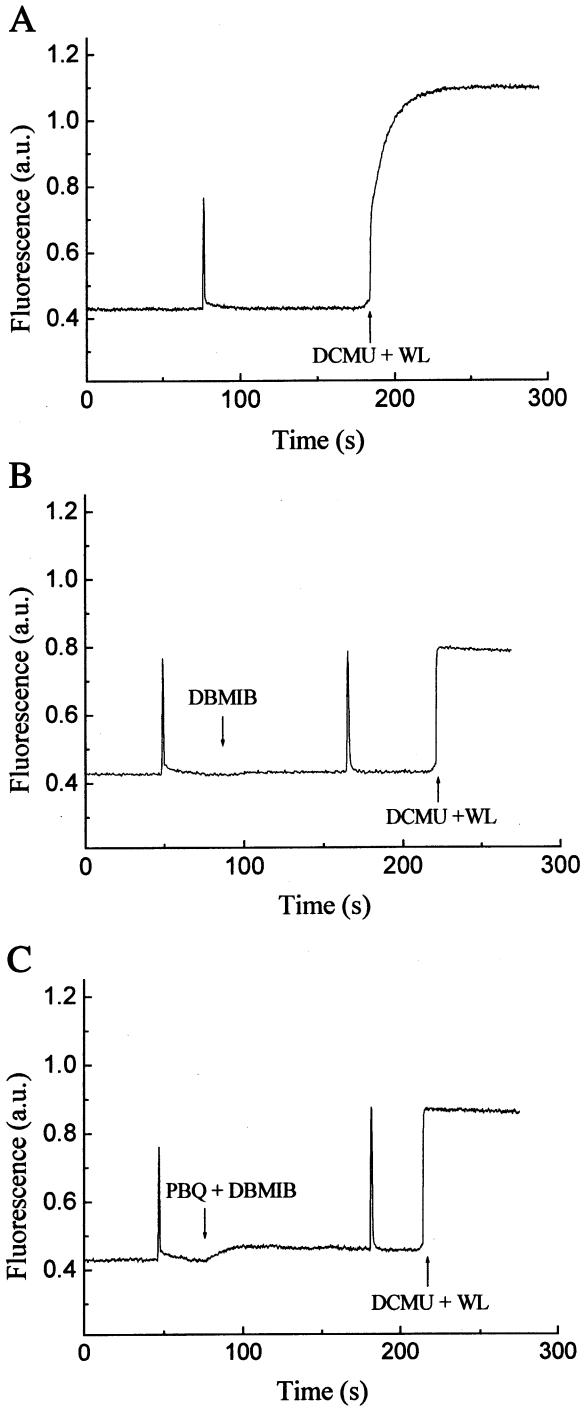

Fig. 4. Fluorescence induction analysis of dark-adapted *Synechocystis* PCC 6803 treated with DCMU and quinone analogues. The final concentration of DCMU was 10 μM. A: Control, no quinone addition; B: 50 μM DBMIB; C: 50 μM PBQ and 5 μM DBMIB. Arrows indicated the additions of various reagents. WL, white light (200 μE m^{-2} s^{-1}).

(or cytochrome c_{553}) were completely oxidized by PS I activity in the presence of DCMU and actinic light (Fig. 4A). Since DBMIB blocked the transfer of electrons from the PQ pool to the cytochrome b_6f complex, these electron carriers were oxidized by respiratory chain terminal oxidases in DBMIB-treated cells which remained in state 2.

So it seems that the redox states of all these carriers were irrespective of state transitions in cyanobacteria. However, DBMIB could prevent PQ from binding to the Qo site of the cytochrome b_6f and interrupt the relation between the redox state of the PQ pool and state transitions. Therefore we propose that both PQ and the cytochrome b_6f are involved in state transitions in vivo. According to our results, it is obvious that PQH_2 induced a state 2 transition whereas PQ caused the opposite effect.

Mullineaux and Allen found that the presence of duroquinol caused a transition to state 2 in *Synechococcus* 6301 [19]. It was reported that duroquinol did not serve as a reductant of the PQ pool, but directly donated its electrons to the cytochrome b_6f complex by binding to the Qo site [37]. These facts also imply that the cytochrome b_6f is involved in state transitions in cyanobacteria.

In chloroplasts, LHC II kinase is activated when PQH_2 occupies the Qo site of the cytochrome b_6f [14]. According to the models of cytochrome bc_1 complexes [38–40], occupancy of the Qo site by ubiquinol drives Rieske protein to move to the proximal position close to the heme b_L. When the ubiquinol is oxidized, the Rieske protein moves either to an intermediate position [39] or a distal position close to the heme of cytochrome c_1 [40]. Breyton found that the Rieske protein of the cytochrome b_6f complex underwent a similar kind of large scale movement as in the bc_1 complex during electron transfer from PQH_2 to the heme of the cytochrome *f* subunit by electron crystallography [41]. The conformational change of the Rieske protein is crucial to activate LHC II kinase [12–14,42], and Zito and his colleagues suggested that the LHC II kinase was activated when the Rieske protein was in its proximal position [12].

Schoepp et al. reported that all redox states of DBMIB could strongly interact with the 2Fe–2S cluster of the Rieske protein and lock the Rieske protein in the proximal position [43]. In the present work, both DBMIB and PQH_2 induced a state 2 transition in *Synechocystis* PCC 6803. These results are in agreement with the hypothesis of Zito et al.

Here we propose a possible regulatory mechanism for state transitions in cyanobacteria. The redox state of the PQ pool is pivotal and the conformational changes of the Rieske protein are involved in state transitions. When PQH_2 binds to the Qo site, the Rieske protein moves to the proximal position and then subsequently activates a special signal transduction pathway, which induces a state 2 transition. When the PQ pool is oxidized, this pathway is blocked and state 1 appears. Since the PQ pool is reduced by respiratory electron flow in the dark [17], the typical dark state of carbohydrate-replete cyanobacteria is state 2. This model implies that the regulatory mechanisms for state transitions are conserved to some extent in cyanobacteria, green algae, and higher plants.

According to this model, the effects of exogenous reagents on state transitions can be interpreted as follows: (i) PBQ and DBMIB. The effects of PBQ and DBMIB on state transitions resulted from their effects on the oxidization of the PQ pool, and from their direct interactions with the Qo site of the cytochrome b_6f complex. The occupancy of the Qo site by reduced quinones or DBMIB induced the movement of the Rieske protein to the proximal position and then elicited a state 2 transition. By contrast, the occupancy of the Qo site by oxidized quinones, including PBQ and PQ, would induce a shift to state 1. In the presence of PBQ, the PQ pool was oxidized and most of the Qo sites were occupied by PBQ or

PQ, subsequently a state 1 transition appeared (Figs. 1 and 2). Since DBMIB has a higher affinity to the Qo site, the majority of Rieske proteins were kept in the proximal position and cells would be substantially in state 2 in the presence of the same concentration of DBMIB and PBQ (Fig. 3). (ii) DCMU. Under illumination, DCMU blocked the transfer of electrons from PS II turnover and caused the oxidation of the PQ pool, which induced a transition to state 1. Since DCMU itself was not involved in state transitions, it could not reverse the effects of PBQ and DBMIB (Fig. 4).

It should be noted that DBMIB inhibits state 2 transition by preventing the phosphorylation of PS II and LHC II in chloroplasts [7,14,15]. Finazzi et al. proposed that DBMIB binding also activated LHC II kinase, but it fixed the conformation of Rieske protein in proximal position and therefore prevented cytochrome b_6f from releasing the activated kinase [15]. The fact that DBMIB induced a state 2 transition in *Synechocystis* PCC 6803 indicates some special aspects of the regulatory mechanism for state transitions in cyanobacteria.

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